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# Early-onset Bipolar Disorder: The Relationship Between Symptoms and Family Functioning

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Running Head: EARLY-ONSET BIPOLAR DISORDER

Early-onset Bipolar Disorder:  
The Relationship Between Symptoms and Family Functioning

A Honors Thesis Presented by  
Meryl Yoches  
To the Department of Psychology  
In partial fulfillment of the requirements  
for a Bachelor of Arts Degree

Connecticut College

New London, CT

April 28<sup>th</sup> 2006

## Abstract

The incidence, course, and diagnostic criteria of early-onset bipolar disorder are heavily debated within the psychological community. Although new research has solved some of the uncertainties about the disorder, questions remain about its course, presentation and specific features in childhood. The goal of this study was to evaluate the relationship between family functioning and symptom presentation. The cases of 406 children diagnosed with bipolar disorder were examined. The statistics suggest that not only are there differences in symptom presentation among bipolar type, sex and age, there are also differences in family functioning. Conflict and cohesion level appear to be the most related to symptom presentation in children, but especially related to manic

## Acknowledgments

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## Early-Onset Bipolar Disorder:

### The Relationship Between Symptoms and Family Functioning

Through the past ten years, much research has been devoted to the understanding of bipolar disorder but disagreement on its diagnosis remains within the psychological community (McClure, Kubiszyn & Kaslow, 2002). Even with new research, questions still remain on its presentation, course and correct diagnosis in young children. Although presently researchers and clinicians accept that bipolar disorder exists in young children, this has not always been the case. Questions about the capacity for children to experience both mania and depression have been debated for many years (McClure, Kubiszyn & Kaslow, 2002).

Originally, researchers thought that the only way children and adolescents could present with bipolar disorder was in the same way adult patients manifested the disorder (Carlson, 2005). Under these strict constraints it was clear that childhood bipolar disorder was very rare and possibly non-existent. Through research with children, however, it became more obvious that some classic symptoms of bipolar disorder were not present in children while others were. More recently, it has been proposed that there may be additional differential classifiers and symptoms in children with bipolar disorder that bear little obvious relationship to the adult illness (Carlson, 2005; NIMH, 2000).

Classic adult bipolar disorder consists of periods of both mania and depression. Manic episodes may include grandiosity, excessive spending and pressured speech, while depressive episodes include down moods, changes in appetite and sleeping patterns. Problems arise in identifying childhood onset bipolar disorder in part because children may not be developmentally able to present with symptoms in the same way that adults

do. Very young children cannot go on wild spending sprees, drive across the country on the spur of the moment or have “classic” grandiose ideas that are common characteristics of adult mania. Similarly, children may not be able to verbally express the sadness they feel while in a depressive episode in the same way as adults; instead they may show their symptoms behaviorally and somatically (Werner & Kerig, 2000).

The concept of “adulthood morphism,” or the assumption that a disorder will present itself in the same way in adults and children, is becoming somewhat obsolete with bipolar disorder (Werner & Kerig, 2000). This is increasingly the case with other psychological disorders in childhood as well. Similarly, the concept of “heterotypic continuity,” or the presentation of the same underlying disorder through different behavioral symptoms during different time periods across the life span, (Cicchetti & Rogosch, 2002) has become increasingly helpful in our understanding of bipolar disorder over the lifespan. Research is ongoing to understand both the consistencies and inconsistencies in the behavioral display across developmental stages in order to advance this knowledge and find out more about the course of the disorder.

This literature review will explore many of the relevant issues that researchers are investigating today. It will begin by examining the history of bipolar disorder diagnosis in children and explore the differences between adult and child bipolar disorder. Topics including differential diagnoses for children as well as current causal theories and treatment options will also be investigated. This literature review will also lay the groundwork for the current investigation, which uses data from an ongoing longitudinal study of childhood bipolar disorder. The data was collected using state of the art diagnostic criteria reflecting our current understanding of the disorder to examine several

aspects of childhood bipolar disorder. Descriptive analyses of bipolar type, sex and age differences in symptom presentation will be examined. Additionally, the relationship between symptom severity and family functioning will be explored. Hopefully, this examination will add to the growing body of literature about this under-researched disorder.

### *Historical Background*

To appreciate current conceptualizations of bipolar disorder and the controversy surrounding its diagnosis in children, it is important to review the history of research and theorizing about bipolar disorder in childhood. Four hundred years ago adults suffering from bipolar disorder were deemed “insane” and clumped in a large group with people suffering from other severe mental disorders (Torry & Knable, 2002). More recently, adult bipolar disorder was distinguished from other mental illnesses, and the possibility of the disorder affecting youth was introduced. John Haslam’s *Observations of Madness and Melancholy* (1809) was the first to describe children with mania. These children were described as getting “little sleep, [as being] loquacious and disposed to harangue, and [to] decide upon every subject that may be started,” (Torry & Knable, 2002, p. 11).

More modern cases of mania in children were recorded starting in the twentieth century (Kraepelin, 1921; Kasanin, 1931). Kraepelin (1921) is credited with the modern identification of bipolar disorder in adults, as well as describing how the disorder affects children. He portrayed children in manic episodes as assertive and grandiose, the same descriptions used for adults. Contradicting Kraepelin, Kasanin (1931) suggested that the adult classification system used to diagnose children may be flawed for this purpose. He

noted behavioral differences between adults and children through case studies of ten bipolar children.

Later, in 1950, the question of early-onset bipolar disorder was raised in the book *The Nervous Child*, but it was described as being very rare (Carlson, 2005). During the late 1970s, published reports on different symptom presentation in children with bipolar disorder compared to adults began to appear. Weinberg and Brumback (1976) published a modification to the symptoms outlined in the DSM-III specifically for children. In their manual, criteria for manic episodes included euphoria and irritable moods as central symptoms, as well as hyperactivity, “push of speech,” flight of ideas, grandiosity, sleep disturbance and distractibility as additional symptoms. They based these criteria on their own observations of children. This symptom checklist was quickly dismissed, however, because it was said to describe hyperactive children and not manic children. Confusion still remains in differentiating the symptoms of children who are exhibiting hyperactivity from those experiencing a manic episode (Sanchez, Hagio, Weller, & Weller, 1999).

Just as with mania, depression in children has been recently recognized as presenting itself differently from adult depression. Cameron (1924) was one of the first to record the occurrence of depression in children. He observed their sad disposition but the severity of these moods was not taken seriously. He suggested that parents could bring their child’s mood up if they treated him or her normally and did not do anything out of the ordinary to brighten moods. Cameron believed that these “sad moods” in children were similar to the adult ebb and flow of mood.

In this way depression was also assumed to be adultomorphic, but subsequent research and clinicians’ observations revealed several age-related differences in

depressive symptoms in children compared to adults. Only in the second half of the twentieth century was childhood depression recognized as legitimate and serious, and as being expressed differently in children. One key difference observed was the presence of increased restlessness and irritability in children. Other symptom differences include more behavioral problems in younger children versus more cognitive difficulties in older children (Wenar & Kerig, 2000).

Toward the end of the 1970s and into the 1980s, bipolar disorder in children, along with other under-investigated childhood disorders, began to be researched in a different way, through empirical study rather than by case reports. This provided more insight into the entire population of affected youth. Many published reports featured small sample descriptive studies of patients diagnosed before puberty. During this period, the possibility that pre-pubescent bipolar disorder may be a unique disorder in the bipolar disorder spectrum was raised (Ballenger, Reus, & Post, 1982; Bashir, Russell & Johnson, 1987). Additionally, the problem of misdiagnosis was brought to the forefront of questions being raised about the disorder. Attention to behavioral problems and comorbidity in potentially bipolar youth were also central to research in children (Ballenger, Reus, & Post, 1982; Hassanyeh & Davison, 1980; Werry, McClellan & Chard, 1991). More recent research has advanced beyond these small sample descriptive studies and has focused on both treatment studies and large scale longitudinal studies of the course of childhood bipolar disorder.

Although currently bipolar disorder is recognized as occurring in children, there are still no specific diagnostic criteria for pre-pubescent children. Many researchers continue to advocate for the development of modified diagnostic criteria for children.

Even the current Diagnostic and Statistical Manual, 4<sup>th</sup> edition, Text Revision (DSM-IV-TR) continues to apply adult criteria for the childhood disorder (Sanchez, et al., 1999).

### *Adult Bipolar Disorder*

In order to understand childhood bipolar disorder fully it is important to understand how DSM-IV-TR classifies the adult illness and how these diagnostic criteria do and do not describe the childhood manifestations that have been observed. Between 1 and 1.5% of the general population suffers from bipolar disorder, affecting equal numbers of males and females (Comer, 2004; American Psychiatric Association [APA], 2000). Although Bipolar I is slightly more common than Bipolar II, the number of individuals affected is still relatively low. The typical age of onset for bipolar disorder is anywhere from late adolescence until mid life, with most patients reporting onset in their late teens to early twenties (APA, 2000).

According to the DSM-IV-TR, to meet diagnostic criteria for Bipolar I disorder a person must have at least one manic episode. This is characterized by an “abnormally high or elevated mood” for at least one week at a time, and several other mood symptoms that cause significant disturbance in the person’s life. These symptoms may include some, but not all of: “inflated self-esteem, decreased need for sleep, pressured speech, flight of ideas, distractibility, increase in goal directed activity [and] increased involvement in pleasurable activities that have a high potential for painful consequences” (APA, 2000, p. 362). There does not have to be a history of major depressive episodes, although often there is. A major depressive episode consists of a period of at least two weeks of depressed mood or anhedonia, which is a loss of interest or pleasure in most or all activities. At least five other mood symptoms must co-occur during this time,

including: weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feelings of worthlessness, diminished ability to think or concentrate and recurrent thoughts of death or suicide attempts (APA, 2000). To meet criteria for Bipolar I, one may also experience a mixed episode, which is characterized by symptoms of a manic episode and a major depressive episode within a one week interval. Mixed episodes involve rapidly changing moods and are often times accompanied by: “agitation, insomnia, appetite dysregulation, psychotic features, and suicidal thinking” (APA, 2000, p. 362).

Bipolar II, on the other hand, is characterized by hypomanic episodes, or mild periods of mania, in addition to at least one major depressive episode. A hypomanic episode can be differentiated from a manic episode by both length and severity. Hypomanic episodes are described as a “persistently elevated, expansive or irritable mood, lasting throughout at least 4 days” (APA, 2000, p. 368). The presence of a manic episode or mixed episode rules out the diagnosis of Bipolar II. Finally, Bipolar NOS (Not Otherwise Specified) is diagnosed when bipolar-like symptoms occur together but do not meet full criteria for a manic episode, a hypomanic episode or depressive episodes. This may also include the experience of hypomanic episodes without the presence of major depressive episodes or symptoms that meet criteria for one of the bipolar disorders but do not occur on the correct timeline for diagnosis (APA, 2000).

### *Child Bipolar Disorder*

Although the current DSM-IV-TR can lay the groundwork for diagnosing bipolar disorder in children, it does not take into account many differences in the way symptoms in children may occur compared to adults (Adleman, Barnea-Goraly, & Chang, 2004;

NIMH, 2000). Without a specific manual to diagnose childhood mental disorders, including bipolar disorder, inaccurate diagnosis or misdiagnosis is more likely to occur (Sanchez, et al., 1999). The importance of accurate diagnosis is imperative because if left untreated, bipolar disorder may be less responsive to treatment later on (Lofthouse & Fristad, 2004). Recent research has elaborated on key developmental differences for bipolar disorder.

Bipolar disorder in children is still considered very rare; one percent of youth are thought to suffer from it (Doyle, Wilens, Kwon, Seidman, Faraone, & Freid, 2005; Sanchez, et al., 1999). Many children are first diagnosed with Bipolar NOS because their presentation does not meet criteria for a full manic or depressive episode (McClellan and Werry, 1997). Two of the biggest differences between the presentation of symptoms in children compared to adults are the type and length of mood episodes. Children often have more mixed episodes, experience more rapid cycling and have recurring mood deregulation (Wagner, 2003; Geller & Luby, 1997; Lofthouse & Fristad, 2004; Biederman, Mick, Faraone, Spencer, Wilens, & Wozniak, 2000).

Mania in children may look more like irritability and the externalization of anger rather than the euphoria that characterizes adult mania. Children often engage in reckless behavior, overactivity, hyper-sexuality, psychomotor agitation, distractibility, aggression, poor school performance and restless sleep (Sanchez, et al., 1999). Children also tend to be more irritable in their manic moods compared with older patients (Geller, Zimmerman, Williams, Delbello, Frazier, & Beringer, 2002b; Wozniak, Spencer, Biederman, Kwon, Monuteaux, Rettew & Lail, 2004) and have quick mood changes between irritability and other symptoms during these episodes (Bowring & Kovacs,



1992). The kind of irritability seen in children with mania is very severe and is seen as very distinct from other forms of irritability in other disorders. Researchers in one study concluded that manic children seem “super angry” in open ended interviews compared to participants who were irritable and diagnosed with Attention Deficit Hyperactivity Disorder (Wozniak, Biederman, Kwon, Mick, Faraone, Orlovsky, et al., 2005).

Very young children are more hyperactive, aggressive and more euphoric during their manic states (Carlson, 2005; Biederman, Faraone, Mick, Wozniak, Chen & Ouellette, et al., 1996). The earlier a child presents with symptoms, the more severe their symptoms will generally be (Carlson, 2005). An additional difference that can be seen in children with bipolar disorder is the increased presence of psychotic features compared to adults with bipolar disorder. Especially because children have more mixed episodes, where psychotic features are more common, Ballenger Rues, and Post (1982) found that mania in children presents with psychotic features more than it does in adults.

For depression, children may complain of somatic symptoms, poor school performance, irritability, social isolation and frequent crying (NIMH, 2000). Children and adolescents in a major depressive episode generally view the entire world in negative terms. They use the depressive cognitive triad which looks at the world, self and the future in a negative light (Asarnow, Carlson, & Guthrie, 1987). Although there appears to be differences between child onset and adult onset bipolar disorder, research shows that children with bipolar disorder generally continue to suffer from the disorder into adulthood where they may go on to present with classic symptoms (APA, 2000). This also lends credibility to the definition of heterotypic continuity for children and this disorder (APA, 2000).

An additional way that researchers have begun to study bipolar disorder is by looking at studies utilizing retrospective interviews of adults with bipolar disorder to reveal patterns of symptoms in childhood (Geller & Luby, 1997; Lish, Dime-Meenan, Whybrow, Price & Herschfeld, 1994). Research finds that many patients diagnosed in adulthood report that their mania began in childhood, even if they were never diagnosed until adolescence or adulthood. Geller and Luby (1997) found that 20% to 40% of adults with bipolar disorder reported an onset of symptoms during childhood; similarly, Lish and colleges (1994) found that 59% of participants in their sample reported symptoms in childhood. These studies are somewhat limited because memories may be biased by current diagnosis and adult patients may have subjective recall of symptoms from many years ago. Retrospective interview studies may show a higher number of patients reporting symptoms at a younger age because people may recall symptoms consistent with their present functioning (Frazier, Ahn, DeJong, Bent, Breeze, & Giuliano, 2005).

Egeland, Hostetter, Pauls, and Sussex (2000) utilized a different retrospective technique to address this problem. The researchers examined early symptoms of bipolar adults through patient and family reports of child behavior history taken at first hospitalization, prior to their diagnosis of bipolar disorder. An important factor in this study was that childhood history was gathered before formal diagnosis to minimize the influence of diagnosis on retrospective report. Only behavioral symptoms that were recorded in the hospital records were employed and no checklists or prompting devices were used for the raters collecting the data. Important findings in this study include the episodic nature of mood symptoms in children, changes in energy and sleep disturbances. Additionally, symptoms in these individuals looked more like classic bipolar disorder

with age, providing more evidence for heterotypic continuity. The underlying diathesis of bipolar appears to be expressed differently at different ages.

While pre-adolescent onset bipolar disorder may manifest differently from adult bipolar disorder, some studies have noted that adolescent onset bipolar disorder is very similar to classic adult bipolar disorder (Geller, et al., 2002b). Thus, the clear mood cycling of adult bipolar disorder seems to develop with age, but may have its beginnings in a less differentiated mood disturbance. “The bipolar controversy, then, is not about whether classic manic depression has been missed in children. It is about what a broader definition of mania with less clear cut episodes and more childhood psychopathology and comorbidity represents” (Carlson, 2005, p.355).

*Course and Severity:*

In addition to the clear differences in presentation of symptoms of childhood bipolar disorder compared to adult bipolar disorder, it is also important to note the difference in course and severity of bipolar disorder in childhood compared to adulthood. For example, many reports have shown that early-onset bipolar disorder in children looks very similar to treatment resistant bipolar disorder in adults, especially in its unique presentation and greater probability of mood congruent psychosis (Geller & Luby, 1997). This is important in looking at children with symptoms that seem close to bipolar disorder but may not meet adult criteria. Also, these children may be more resistant to treatment and have a more severe and chronic illness throughout their lives (Geller & Luby, 1997; Lofthouse & Fristad, 2004).

In looking solely at childhood bipolar disorder, not much is known about its course. Retrospective studies looking back on adults currently diagnosed with bipolar

disorder reveal some clues about what the eventual course will look like. It seems as though there are two possible paths this disorder will take in children over time. One path is that children will eventually present with symptoms that look more like classic adult bipolar disorder; the other way children may develop is with the extreme subtype of chronic treatment resistant bipolar disorder (NIMH, 2000). Other diagnostic outcomes are possibly included in this spectrum, such as cyclothymia or borderline personality disorder, but little research has been conducted examining this broad range of disorders. More research is needed to examine the course of this disorder from first symptom presentation until adulthood.

### *Differential Diagnoses*

The importance of clarifying diagnostic criteria for childhood bipolar disorder is especially vital because misdiagnosis can be damaging. Factors specific to bipolar disorder that make it especially hard to diagnose include the low number of children who have the disorder relative to other mental illnesses in children. More importantly, the symptoms of childhood bipolar disorder often overlap with other disorders in childhood (Sanchez, et al., 1999).

*Attention Deficit Hyperactivity Disorder.* Geller, Zimmerman, Williams, DelBello, Bolhofner, and Craney, et al. (2002a) studied bipolar children, children with attention-deficit hyperactivity disorder [ADHD], and control participants without any disorder to examine the symptoms specific to each disorder. The study sought to differentiate the symptoms of bipolar disorder and ADHD because child-onset bipolar is almost always diagnosed comorbidly with ADHD (Asarnow, Tompson, Hamilton, Goldsteini, & Guthrie, 1994). Additionally, the behaviors that meet diagnostic symptom

criteria are similar for the two disorders. The researchers sought to determine whether these two disorders can be seen as one disorder in children with bipolar disorder or whether the dual diagnosis is correct. The study revealed that although both groups of children presented with poor judgment and irritability, there appeared to be five key symptoms that differentiated bipolar disorder from ADHD.

These distinguishing symptoms included: elation, grandiosity, flight of ideas, decreased need for sleep and hyper-sexuality. These symptoms were shown to be central in bipolar disorder but not in ADHD. Other studies have noted that flight of ideas is a good discriminating feature between childhood bipolar and ADHD (Geller, et al. 2002b). This study demonstrates that although there seem to be many common symptoms of childhood bipolar disorder and ADHD, these two disorders are distinct, and the dual diagnosis in many children is probably correct.

Another study analyzed young clinically referred children with mania, children with ADHD without mania and children without ADHD. Significant differences in symptoms were found between these groups of children, including differences in treatment, medication and hospitalization. Manic children were more likely to have previous hospitalizations compared to non-manic children. Additionally, children who met diagnostic criteria for bipolar disorder and ADHD presented separate symptoms for both disorders, suggesting again that both disorders are probably present independently (Wozniak, Biederman, Kiely, Ablon, Faraone, & Mundy, et al., 1995).

In yet another study addressing differential diagnosis, Carlson (1990) noted the major difference between bipolar disorder, ADHD, and other externalizing disorders. The biggest difference noticed is that bipolar disorder is episodic, while the other

disorders are chronic and have an earlier onset, before the age of six or seven. This finding points to the importance of setting up more definite guidelines for diagnosis of bipolar disorder in children, especially when connections like these are made between age of onset and disorder. “The further one gets from requiring clear-cut episodes of disorder, as part of the definition, the more muddled these waters become” (Carlson, 1990, p. 334).

*Schizophrenia.* In addition to the misdiagnosis of bipolar disorder as childhood externalizing disorders, bipolar disorder is also misdiagnosed as schizophrenia. In fact, the most common misdiagnosis of child-onset bipolar disorder is schizophrenia. Some researchers argue that early-onset bipolar disorder is misdiagnosed 50% of the time (McClellan and Werry, 1997). One reason for this problem is that previously it was believed that early-onset schizophrenia was more common than early-onset bipolar disorder so when children came in for diagnosis it was more likely that the symptoms would be seen as schizophrenia (Carlson, 1990). An additional reason for the misdiagnosis as schizophrenia is that psychotic features of mania in young children are often seen as symptoms of schizophrenia. If the child presents with bipolar disorder with hallucinations or delusions, it becomes important to identify when the psychotic symptoms occur to diagnose the child accurately (Joyce, 1994; Ballenger, Reus & Post, 1982; Werry, McClellan & Chard, 1991).

Symptoms that are common in schizophrenia, including delusions, hallucinations, paranoid ideation and catonia have been shown to occur in children with mania after longitudinal follow-up. Some patients diagnosed with “atypical” schizophrenia turn out to have bipolar disorder with psychotic features instead. Ballenger Rues, and Post (1982)

found that mania in children might present with psychotic features more often than it does in adults, which is important to note when examining children presenting with psychosis. Other studies have confirmed this, saying that the earlier the age of onset of bipolar disorder, the more likely the individual will display psychotic features (Joyce, 1994).

Previous research has examined case studies of bipolar disorder with psychosis in order to gain a better picture of how the disorder is specifically different from schizophrenia. Hassanyeh and Davison (1980) reviewed the case histories of 10 bipolar patients with psychosis under the age of 16. They found that the presence of delusions and hallucinations might lead to the suspicion of schizophrenia but that delusions and hallucinations in bipolar disorder are only present during mood episodes. They recommend that attention be paid to the timing of these symptoms in order to distinguish between schizophrenia and bipolar disorder. The researchers also noted that mania was easier to recognize than depression in youth, but mania may be misdiagnosed as behavioral problems associated with adolescence.

Carlson, Fennig, and Bromet (1994) studied children diagnosed with schizophrenia at first admission to a psychiatric hospital and six months later, in order to examine any changes in diagnosis that could indicate misdiagnosis of bipolar disorder as schizophrenia. They used the new edition of the DSM to help clarify diagnoses. They found that within the hospital setting, there was a problem interpreting symptoms, such as psychosis, that are fleeting, as criteria for mania. These authors noted that schizophrenia is not the alternative diagnosis for bipolar disorder since the DSM-IV was published. Instead, psychosis NOS or schizophreniform disorder is often diagnosed when the nature

of the psychotic symptoms are unclear. A further finding reveals no difference in the prevalence of bipolar disorder and schizophrenia in young children. This is important because previous reasons for misdiagnosis of schizophrenia were based upon the assumption that schizophrenia occurred more in children.

### *Causal Theories of Bipolar Disorder*

*Biological Causes.* Research suggests that bipolar disorder is biologically rooted. According to the DSM-IV-TR there is an increased risk of having the disorder if one has a first degree relative with Bipolar I, Bipolar II or Major Depressive Disorder (APA, 2000). This is why it is important to examine a child's family history when determining diagnosis (Coyle, Pine, Charney, Lewis, Nemeroff & Carlson, et al., 2003). Many researchers see knowledge of family history as central to diagnostic decision making. However, not everyone with heavy familial loading for bipolar disorder will develop it, while others without a family history do. Researchers have developed a bio-psycho-social explanation that accounts for this. This model explains that certain people are predisposed to bipolar disorder biologically, but the onset of the disorder may be affected by psychosocial factors such as low maternal child warmth, high parental child tension or poor peer relations (Lofthouse & Fristad, 2004). Another way to look at this is through a biological vulnerability model. In this model biological factors might influence what disease a person is predisposed to but the actual manifestation of the disorder is shaped by other biological or social factors (Johnson & Miller, 1997).

Many studies have tried to pinpoint the precise biological mechanisms of bipolar disorder, but the picture is complex. For example, Geller and Luby (1997) propose that different forms of bipolar disorder, including early-onset, may have different



neurobiological mechanisms. These different mechanisms may affect the severity of the disorder depending on age of onset and development of brain structure.

To better understand the biological bases of child onset bipolar disorder, Frazier and colleagues (2005) performed a meta-analysis reviewing early-onset bipolar disorder studies using MRI scans. In their review of literature, they found that early-onset bipolar disorder is associated with functional and anatomic abnormalities that influence aspects of affect regulation and cognition. They found that young patients with bipolar disorder have increased ventricular white matter as well as decreased amygdala size compared with controls. Additionally, the superior temporal gyrus in bipolar youth has been found in several studies to be significantly smaller than controls. Finally, several studies reviewed noted a smaller hippocampus in bipolar children compared to healthy controls.

In comparing these studies to what has been found in adults, Frazier et al. (2005) argue that there is a definite difference in the brain structures of child bipolar patients compared with adult patients. Studies on adult bipolar patients do not reveal differences in brain structure compared to individuals without bipolar disorder (Frazier, et al., 2005). The authors suggest that bipolar youths may have a neurodevelopmental problem that affects the total cerebral volume and contributes to their disorder. Currently, the studies examining adults do not take into account what proportion of their subjects experienced early-onset bipolar. The proportion may affect why there is no difference. Better clarification of bipolar history will lead to more definitive results in the future.

In another study utilizing MRI scans, Adelman, Barnea, Goral and Chang (2004) reviewed studies examining brain differences between healthy and bipolar youths. Although many studies have discussed an increase in thalamus size in children with

bipolar disorder, others have noted that the thalamus may be smaller in these children compared to children without any psychological disorder. This study found that when thalamus size of bipolar children is compared to schizophrenic patients, there is no statistical difference in size. The authors also explain that adolescents with bipolar disorder have a significantly greater proportion of white matter than those without bipolar disorder. Although much of the data reviewed is new, the authors suggest the possibility of using this information to diagnose children earlier with the aid of MRI scans. Such decisions might be made if the child has a heavy family history or presents with symptoms at an early age. The developmental neuroimaging diagnostics may someday help clarify the diagnosis of bipolar disorder and reduce the chances of misdiagnosis. Unfortunately, MRI studies are limited in number. More research must be done in order to continue examining the biological underpinnings of this disorder.

*Social Causes.* The significance of the family environment in mental illness is especially important with children because children are embedded in the family environment more than adults. The family shapes the way a child sees the world and may be a source of both support and stress, depending on the child. Factors such as the daily strain of having a parent with mental illness can create more disorganization at home and engender more family conflict. These are important variables to examine when trying to understand childhood mania and depression. It is important to study the interplay of biological and family factors with bipolar disorder because parental mental illness is common and it presents both a biological and a social risk to the child.

As the bio-psychosocial model states, biology is not the sole cause by which one develops bipolar disorder. Johnson and Miller (1997) studied bipolar patients to examine

how negative life events influenced their symptom severity. They found that individuals who experienced major stressors in their lives after the onset of the disorder took longer to recover than those patients without psychosocial stress. The authors also found that part of the stress that influences the outbreak of a manic or depressive episode may also relate to how their family views their disorder. Those who reported having families who believe the patient has control over their symptoms may not respond to the stress in the patient's life in a supportive way. Other studies examining life stress reveal that bipolar individuals with more stress tend to relapse more than those with less stressful family milieus (Rea, Tompson, Miklowitz, Goldstein, Hwang, & Mintz, 2003). For individuals with bipolar disorder, high levels of family stress have been associated with medication non-compliance as well. Many studies have demonstrated that patients returning to stressful home lives after hospitalization relapse more often and more quickly than patients returning to less stressful homes (Rea, et al.; 2003, Holahan & Moos, 1987). The psychological effects of the environment are sometimes just as important as the biological risk factors.

Utilizing archival methods, Brown, McBride, Bauer, and Williford (2005) studied adults with early-onset bipolar disorder and examined their family environments. The researchers inspected an extreme form of stress: childhood history of abuse, including physical, sexual and both. They examined age of onset of bipolar disorder, the severity of illness, and comorbidities. The study revealed that bipolar patients abused as children did not have an earlier age of onset than non-abused patients, but abused patients were more than two times as likely to be involuntarily admitted to a psychiatric hospital. They were also three times more likely to have a comorbid disorder, specifically Post

Traumatic Stress Disorder (PTSD) and two times more likely to have an alcohol use disorder. Additionally, patients with rapid cycling as adults tended to have an earlier age of onset, which is a characteristic of episodes in children. Although the extreme form of stress examined was not related to age of bipolar onset, it was related to distress, impairment and the presence of comorbid conditions suggesting that stress is related to the development and certainly the severity of bipolar disorder.

Another way that stress and bipolar disorder have been examined is through “expressed emotion.” Expressed emotion is a measure of critical statements and over-involvement of family members in one another’s lives (Asarnow, Tompson, Hamilton, Goldstein & Guthrie, 1994). When looking at expressed emotion in families, research reveals that those with higher levels of expressed emotion also have more stress. Within families where expressed emotion and stress are low, bipolar patients have a lower risk of re-hospitalization (Miklowitz, Goldstein, Nuechterlein, Snyder & Mintz, 1988).

Since there is limited data exploring the relationship between family environment and bipolar disorder, studies examining other childhood mental illnesses can be useful to draw connections between family stress and psychopathology. Connell and Goldman (2002) conducted a meta-analysis of studies examining the relationship between internalizing and externalizing behaviors and parental mental illness. This study took special note to record both paternal and maternal mental illness, which is unique because previous studies focused mainly on mothers. Internalizing disorders in this study included anxiety and depression, while externalizing disorders included oppositional defiant disorder, conduct disorder and ADHD. Overall the researchers found no difference in the extent to which externalizing problems were related to maternal versus paternal

mental illness. However, they found that maternal depression was more strongly linked to internalizing disorders in offspring. Additionally, alcoholism and substance abuse in mothers was more closely related to externalizing disorders than to internalizing disorders in children. Alcohol and substance abuse in fathers were not related to childhood symptoms.

Although the relationships found in this research are important, there are some questions as to the bidirectional nature of this relationship. As much as the parents' behavior affects the children, the child's behavior may affect the parental presentation of illness. Despite the direction, there is a relationship between parental mental illness and child psychopathology, which is supported by other studies and needs to be further examined. These other studies reveal relationships between parental dysfunction, maternal risk factors and family support which are significantly linked to distress in children (Gershon, Hamovit, Guroff & Nurnberger, 1987). Furthermore, other research shows that family support provides strong resistance to relapse in children with mental illness (Holahan & Moos, 1987).

Sometimes it is difficult to disentangle family factors from biology because most children are raised with their biological families, which contribute both to the biological and social influences of the disorder. Hammen, Shih and Brennan (2004) examined family processes that might explain the intergenerational transmission of depression from grandmothers to granddaughters. They examined current depressive symptoms in fifteen year old girls along with their relationships with their families. They also examined familial stressors in the context of two generations of maternal depression. The researchers found that the maternal grandmother's depression may affect her daughter's

depression, which in turn may affect the child's depression. The relationship is explained through the creation of a stressful environment brought on because of the disorder. The authors argued that the environment exacerbates the biological risk already present. This study demonstrated that in addition to maternal depression being a risk factor for depression, it can also be traced through multiple generations. Additionally, the transmission may occur at least in part through non-psychological factors including chronic interpersonal stress and impaired parenting. Although the study did not focus on bipolar disorder, connections can be made between the disorders. It is possible that this same intergenerational influence can affect bipolar youth in the same way.

Goodman, Adamson, Riniti and Cole (1994) continued to look at maternal depression and its relationship to child mental illness. They examined the relationship between maternal depression and negative appraisals of their children and their effects on the severity of the child's illness. They found that maternal depression was significantly related to negative appraisals of children. There was also a significant relationship between childhood psychopathology and mother's history of depression. Children of depressed mothers were found to be also more at risk for low self-esteem, related in some part to their mother's negative attitudes toward them. Although no causal links are drawn in this study, it continues to lend support to the relationship between child psychopathology and maternal mental illness.

Fristad and Clayton (1991) assessed the relationship between family dysfunction and the history of psychopathology in 100 psychiatrically disturbed children. The study revealed that family history of illness and dysfunction were related. Children with mood disorders were more likely to have mothers with psychopathology. Children with

behavioral disorders, on the other hand, were more likely to have extended families with psychopathology as opposed to their immediate families. This study could be interpreted, especially looking at the behavioral disorder finding, as purely biological transmission. However, in adding the stress of living with a mother with psychopathology, the possibility of social stress adding to the development of the disorder in children is likely. This provides support for the bio-psycho-social model.

Expressed emotion is an important factor that can be examined in children with other forms of psychopathology. Research reveals that expressed emotion seems to be a non-specific factor for mental illness. In general, parents of children with psychological disorders exhibit more expressed emotion and fewer positive remarks toward their children compared with control families without psychopathology. Additionally, research has shown that increased levels of expressed emotion increases risk of relapse across a wide range of disorders. Those who live in a home with high expressed emotion are five times more likely to have a mental illness (Miklowitz, et al., 1988). In terms of symptoms, high levels of expressed emotion have been shown to predict child externalizing symptoms and impairment over time (Nelson, Hammern, Brennan, & Ullman, 2003).

In a recent study, expressed emotion levels were examined in relation to childhood depression. Compared to controls that were not diagnosed with any disorder, depressed youths were more likely to experience high expressed emotion at home. Compared to youths with attention deficit/hyperactivity disorder (ADHD), depressed individuals were more likely to have mothers who used expressed emotion. There was

no difference in levels of expressed emotion between the control and the ADHD groups (Asarnow, Tompson, Woo & Cantwell, 2001).

Other studies examining depression reveal that when depression is comorbid with other disruptive disorders, including ADHD or conduct disorder there is a significantly higher rate of expressed emotion compared to control subjects without depression (Asarnow, Tompson, Hamilton, Goldsten & Guthrie, 1994). Especially with externalizing disorders, families tend to attribute the child's problem to his or her personality rather than the illness itself. This may account for some of the tension within these families because they may not completely believe in the symptoms of their child's illness (Brewin, MacCarthy, Duda, and Vaughn, 1991). Although these factors are not directly related to specific child pathology there may be more specific links that has not yet been adequately examined and may be found (Kershner, Cohen & Coyne, 1996).

### *Treatment*

“Medications are the single most important aspect of the treatment of manic-depressive illness” (Torry & Knable, 2002, p. 137). Although the social and environmental risk factors in bipolar disorder are being explored, the most common treatment option usually includes medication because of the overwhelming effectiveness in treatment with adults. Although most bipolar medication is not specifically approved for youth, mood stabilizers are commonly prescribed in children and generally thought to be helpful. Different psychological therapy techniques have been incorporated into treatment for bipolar children, but medication is usually the first line of defense (Torry & Knable, 2002).



Since research has shown that the family environment affects symptom presentation of bipolar disorder in children, and child symptoms affect family relations, it has become increasingly important to integrate family therapy into treatment for bipolar disorder. In the past, the primary form of adjunctive psychological treatment was individual therapy. Increasingly, family therapy methods are being researched and developed to determine treatment efficacy and effectiveness with families of bipolar youths since most clinicians working with bipolar youth have come to see family support as a strong protective factor for a child's psychological health (Holahan & Moos, 1987).

Studies on psychoeducational aspects of treatment have shown that the etiological beliefs of family members have an effect on symptom presentation. When family members believe that a child's disorder is not biologically caused, but is instead caused by the patient's internal personality, patients are more likely to relapse. Additionally, the lack of knowledge by families about the mechanisms for the disorder in children is one of the biggest barriers for children and families (MacKinaw-Koons & Fristad, 2004). Because of this, many practitioners believe that a main goal of family therapy should be educating the family on the etiology and course of bipolar disorder. A related goal is to teach them how family dynamics can impact their child's illness (Johnson, Cournoyer, Fisher, McQuillan, Moriarty, & Richart, et al., 2000). Psychoeducational treatment options focus on facilitating the modification of potentially damaging beliefs and attitudes expressed by families toward their children about their disorders (Kershner, Cohen & Coyne, 1996).

Such family focused treatment generally has six goals (Miklowitz & Goldstein, 1997). Goal one includes assisting the patient and his/her relatives in integrating the

experiences associated with episodes of bipolar disorder. Families generally have difficulty understanding the disorder and accepting its seriousness. This part of treatment tries to develop their understanding of the disorder more directly. Goal two includes assisting the patient and his/her relatives in accepting the possibility of future episodes. A third goal of treatment is assisting the patient and relatives in developing a plan for medication management and controlling symptoms that the family can support.

The fourth goal of treatment includes assisting the patient and relatives in distinguishing between the patient's personality and his or her disorder. This helps reduce blame. The fifth goal is to help the patient and family recognize and learn to cope with stressful life events that can trigger relapses and educating them about how they can reduce harmful statements. Finally, the sixth goal is to help families reshape relationships after another episode. The focus of the entire therapy is on communication skills and reducing critical statements.

This treatment tries to focus on the effects of the family environment on the individual with bipolar disorder. Expressed Emotion is examined to see how critical family members are toward one another, and to examine the effects of expressed emotion on patient's mood episodes. Finally, life events stress is examined, specifically because periods prior to manic or depressive episodes have been shown to contain more stressful life events than other periods in a patient's life (Miklowitz & Goldstein, 1997). In addition to creating awareness and communication about the disorder, family therapy provides an opportunity for family members to cooperate in a situation they might otherwise not be able to.

New treatments are continuing to be developed in order to address the difficult dynamics of childhood bipolar disorder. A new method of treatment that has been developed for bipolar children and their families is entitled “naming the enemy.” This treatment emphasizes the child’s positive enduring traits, which is encouraging for the child who is struggling with self-concept and self-esteem because of bipolar disorder. Negative mood symptoms are also discussed, but it is accepted that they are not part of the child, but rather symptoms of the disorder. This treatment can deliver a message of hope for the child and the child’s family (Fristad, Gavazzi & Soldano, 1999).

Fristad, Gavazzi, and Soldano (1999) examined the effect of psychoeducational treatment in children and families with mood disorders. They used standard psychoeducational practices, but adapted them slightly. They investigated three formats for treatment: group workshops, six individual outpatient group sessions for parents and children, and individual family therapy. Results revealed that integrating psychoeducation into these groups had positive effects on teaching families about their child’s disorder and ways about to help the child adjust. Unfortunately no control group was assigned to examine these gains in regards to no treatment, but this does not take away from the effectiveness of the treatment.

In another study, Rea, et al., (2003) studied the difference in treatment outcome for family-focused treatment versus individual treatment for adults with bipolar disorder. The family-focused treatment was based on standard psychoeducation about bipolar disorder, communication enhancement training and the teaching of problem solving skills. Individual therapy was supportive, problem-focused and educational for the patient. The study found that although there was no difference in the probability of

suffering from subsequent episodes depending on therapy, there was a higher probability of relapse rates over time and re-hospitalization for the individual therapy group. The individuals in the family-focused group were also more compliant with their medication after the study. The family focus group was also more compliant with their medication after the study. The researchers concluded that outpatient family therapy can reduce the risk of relapse and re-hospitalization compared to individual therapy. The researchers therefore recommended psychoeducational treatment with the family as an important component in comprehensive outpatient management of bipolar disorder.

#### *NIMH Roundtable Discussion*

Due to the continuing controversy surrounding early-onset bipolar disorder, the National Institute of Mental Health [NIMH] conducted a research Roundtable on the disorder in April of 2000. The NIMH Developmental Psychopathology and Prevention Research Branch, in conjunction with the Child and Adolescent Treatment and Preventative Intervention Research Branch held the Roundtable along with noted researchers and experts in the field. The discussion focused on clinical assessment and treatment for childhood bipolar disorder as well as developing a better definition for diagnosis in children.

The members of the group agreed that the DSM-IV-TR criterion is useful in the diagnosis of bipolar disorder, but they redefined some diagnostic criteria for Bipolar I and II. They stated that although the DSM-IV-TR can lay the groundwork for a diagnosis, in childhood, frequently the disorder includes long duration, rapid cycling and mixed episodes which are not mentioned in the DSM-IV-TR. Bipolar NOS, on the other hand, was discussed as having a chronic and continuous course. Episodes are seen as mainly

irritable and aggressive. The panel agreed that Bipolar NOS is still a good working diagnosis for children as long as attention is paid to the possible confusion with other disorders that have similar symptoms including Attention Deficit Hyper Activity Disorder, Anxiety Disorders, Oppositional Defiant Disorder and pre-pubertal onset Substance Abuse.

The meeting concluded with the agreement that bipolar disorder occurs in prepubertal children. It also concluded that careful analysis and evaluation must be given to children presenting with possible symptoms of bipolar disorder in order to diagnosis the disorder accurately and rule out other conditions in children. This research roundtable was convened to help the research and clinical community make sense of the growing body of literature on childhood bipolar disorder. Participants agreed that much more needs to be known about this still controversial diagnosis. To best answer these questions, large scale studies of children diagnosed using the most current understanding of childhood bipolar disorder are needed. The current investigation attempted to examine key questions about family factors associated with symptom presentation of bipolar youth using data from an ongoing longitudinal study of bipolar youth.

### *Hypothesis*

The current study first aimed to describe the clinical presentation of bipolar disorder in children and adolescents, using a sample diagnosed based on the best current understanding of bipolar symptomatology. The prevalence of Bipolar I/II versus Bipolar NOS were be examined, as were several other variables including medication and treatment history. Symptom differences were then be explored for the different bipolar types and for sex and pubertal status. Several differences were expected. Specifically,

children with Bipolar I/II were expected to present with more mania and more depression than children with Bipolar NOS. Additionally, Bipolar NOS children were expected to have lower global functioning. Since there is very little research on sex differences, differences based on sex are difficult to predict. However, related research has noted that girls are often more symptomatic when diagnosed in childhood (Silverthorne & Frick, 1999); therefore girls were expected to have higher symptom severity scores for depression and also lower scores for global functioning. Finally, differences are expected for pubertal status. Bipolar disorder in adolescents has been observed to be more similar to adult bipolar disorder than has bipolar disorder in children (NIMH, 2000). Thus, post-pubertal children were expected to present with more manic and depressive symptoms than pre-pubertal children, who were expected to have more diffuse symptoms and lower global functioning.

The second main goal of the study was to examine the relationship between family functioning and symptom severity in children and adolescents with bipolar disorder. Based on research with adults (Rea, et al., 2003), a relationship was expected. Specifically, higher family conflict should be related to higher levels of mania and depression. The relationship between nonconflictual family functioning and symptom severity was also explored. There is little research on which to base these predictions. It is possible that less cohesion and less adaptability in families would also be related to higher symptom levels. These relations between family functioning and symptom severity may differ depending on the pubertal status of the child, and this was also explored. Because younger children are more embedded in family life than adolescents,

family functioning is expected to be a stronger predictor of mania, depression, and overall functioning for pre-pubertal children compared to adolescents.

## Method

The present study is an archival investigation of the first wave of longitudinal data from a multi-site study on child onset bipolar disorder.

### *Participants*

Participants were 406 children and adolescents, of whom 217 (53.6%) were male and 188 (46.6%) were female. They ranged in age from 7 years through 18 years with a mean age of 12.72 ( $SD = 3.19$ ). There were 338 participants (83.5%) who classified their race as white and 67 (16.5%) who classified themselves as non-white. Three-hundred eighty five (95.1%) classified themselves as non-Hispanic, while 20 (4.9%) classified themselves as Hispanic. All of the participants currently had a diagnosis of Bipolar I, Bipolar II or Bipolar NOS. The participants were recruited into the COBY (Course and Outcome of Bipolar Youth) study at three sites, Brown University, University of Pittsburgh and University California at Los Angeles. Participants were either referred to the study by physicians or were self-referred through advertisements. They were evaluated at intake to confirm their diagnosis, and were followed every six months to track symptom severity, psychosocial functioning, treatment and family functioning. Parents were interviewed during each interval to measure child symptoms and assess family functioning.

### *Procedure*

Since intakes were done throughout a four year period, follow-up interviews were not yet available on all patients. Therefore, data was only analyzed from the intake interview. Each interview took approximately two hours to complete. During the interview, background demographic information was obtained as well as current



symptoms and general psychosocial functioning. For children aged 7 through 12, parents and children were interviewed together in the same room. For children aged 13 through 18, children and parents were interviewed separately but given the same interview. Parent and child reports were obtained for reliability and to gather multiple perspectives on the child's symptoms and adjustment. Before the interview several self-report measures were sent to the family and were filled out by both parents and the child.

All participants signed informed consent documents prior to entering into the COBY study. They had the risks and benefits explained to them as well as their rights while in the study. The parents and children were told that their consent or refusal to participate in the study would not influence their ability to receive care at any of the hospitals associated with the study, and that they could withdraw at any time. A federal Certificate of Confidentiality was obtained for the study from the U.S. Department of Health and Human Services (DHHS) to further protect participants' privacy.

### *Measures*

The following measures were selected from the larger set of measures used in the COBY project to answer the specific questions outlined in the introduction.

*Kiddie Schedule for Affective Disorders and Schizophrenia for School Age Children, Present and Life Version (K-SADS-PL):* This is a semi-structured interview that records present symptoms and symptom history, relying on the DSM-IV-TR criteria. Sample questions and criteria are available to help rate the symptoms. From this measure, diagnosis of Bipolar I, II or NOS was determined. The K-SADS MRS (Mania Rating Scale) was also used to assess manic symptoms and the K-SADS-Dep-P

(Depression Scale) was used to evaluate depressive symptoms in the present episode (Kaufman, Birmaher, Brent, Rao, Flynn, Moreci, Williamson & Ryan, 1997).

*Kiddie Mania Rating Scale (K-SADS-MRS)*: This instrument is a 21-item, semi-structured interview to assess the current symptoms of a manic or hypomanic episode. It is based on the KSADS-P 4<sup>th</sup> Revision (Kaufman, et al., 1997) and includes some items from the WASH-U-KSADS. For each item endorsement, intensity, frequency, duration and impairment were assessed. A likert scale was used to rate each symptom from 1 (not present) to 6 (extreme, usually resulting in hospitalization). The mania rating scale has been shown to be a reliable measure of symptom severity with a Cronbach's alpha = .0.94 and inter-rater reliability = 0.97 between two raters (Chambers, Puig-Antich, Hirsch, Paez, Ambrosini, Tabrizi, & Davies, 1985). (see Appendix A)

*Depression Scale (Dep-P)*: This is a 30 question, semi-structured interview that was used to assess current depressive symptoms. Each symptom is assessed for intensity, frequency, duration and impairment. A likert-type scale is used to rate each symptom from 1 (not present) to 6 (extreme, usually resulting in hospitalization). The depression scale has been shown to be a reliable measure of symptom severity with a Cronbach's alpha of .0.94 and inter-rater reliability of 0.97 between two raters (Chambers, et al., 1985). (see Appendix B)

*The Children's Global Assessment Scale (C-GAS)*: This is a scale ranging from 1-100 that assesses overall level of functioning of a child or adolescent. It was adapted from the Global Social Adjustment Scale (GAS). It has been shown to have good reliability in addition to discriminant and concurrent validity (Shaffer, Gould, Brasic, Ambrosini, Fisher, Bird, & Aluwahlia, 1983). (see Appendix C)

*The Conflict Behavior Questionnaire (CBQ)*: This is a self-report scale that was completed by both parents and the child and measures conflict between adolescent and parent within the last two weeks. It is a 20-question measure that yields three scores, parent report of conflict behavior in child, child report of conflict behavior in mother and child report of conflict behavior in father. The likert-type questions cover topics ranging from arguments to communication skills. For the purpose of this study, the scores of the three scales were averaged to get one score describing overall conflict in families. The CBQ is a reliable and valid measure of conflict with a cronbach's alpha of .96 (Birmaher, Brent, Kolko, Baugher, Bridge, & Holder, et al., 2000; Grace, Kelly, & McCain, 1993). (see Appendix D)

*The Family Adaptability and Cohesion Evaluation Scales-II (FACES-II)*: This is a 30-item self report measure that measures family adaptability and cohesion. A likert scale is used to rate each question. The scale ranges from 1 (almost never) to 5 (almost always). Separate linear scores are obtained for each scale and families can be divided into types based on scores. Based on their cohesiveness score, families can be divided into disengaged (very low) separated, connected and very connected (very high). Based on their adaptability score, families can be divided into: rigid (very low), structured, flexible, and very flexible (very high). Family categories are developed based on empirically determining family type by raw score. For example, the raw score of 64 on the cohesion scale would be classified in "connected" family type but the same score on the adaptability scale would merit a "very flexible" family type. The FACES-II is a reliable and valid measure of family functioning with a Cronbach's alpha = .87 (Olson, McCubbin, Barnes, Larsen, Muxen & Wilson, 1982). (see Appendix E)

## Results

### *Descriptive Analyses*

First, descriptive analysis of diagnosis type, episode status, medication history and status, and hospitalization history are provided as an overview. The diagnoses of Bipolar I and Bipolar II were collapsed into one group and compared with Bipolar NOS in another because most research has focused on the difference between the formal diagnoses of Bipolar I or II and the more un-defined diagnosis of Bipolar NOS. The majority of the participants were diagnosed with Bipolar I or II, 264 (65.2%), with the rest being diagnosed with Bipolar NOS, 140 (34.6%). The bulk of the participants were currently in an episode, 262 (64.7%); 85 (21%) were in partial remission and 56 (13.8%) were recovered. Most of the participants were also currently on medication, ( $n = 347$ , 85.7% versus  $n = 56$ , 13.8% not on medication). In terms of past medication, most of the participants were on medication at one time, ( $n = 377$ , 93.1% versus  $n = 28$ , 6.9% never on medication). Two-hundred nineteen children (54.2%) had been in a psychiatric hospital at one point, while 185 (45.7%) had not.

Next, Mania Rating Scores were correlated with Depression Scores and Children's Global Assessment of Functioning to examine the interrelatedness of the different symptom scales used as dependent variables in subsequent analyses. Each of the scales was at least modestly correlated with one another (Table 1), with mania symptom severity being more strongly correlated with overall functioning than depression. Mania Severity was correlated with Depression severity and both were negatively correlated with Current CGAS. This suggests that with greater symptom severity, there is a lower overall functioning.

Table 1

*Intercorrelations Between Symptom Measures*

	<b>Current MRS</b>	<b>Current Dep</b>	<b>Current CGAS</b>
Current MRS	---	.33**	-.43**
Current Dep		---	-.28**
Current CGAS			---

\* Correlation is significant at the .05 level (2-tailed)

\*\*Correlation is significant at the .01 level (2-tailed)

MRS = Mania Rating Scale; Dep = Depression Scale; CGAS = Children's Global Adjustment Scale

### *Differences by Bipolar Type*

Type of bipolar diagnosis (I/II vs. NOS) was examined first. Next, a series of between group comparisons were performed using t-tests to examine differences in symptom presentation between children diagnosed with Bipolar I/II versus those diagnosed with Bipolar NOS. Current mania symptom severity, as measured by the K-SADS Mania Rating Scale, depression symptom severity, as measured by the K-SADS Children's Depression Inventory, and overall level of functioning, measured by the Children's Global Assessment Scale, were indices of symptom presentation. Neither, Mania rating scores,  $t(396) = 1.82, p = .07$ , nor depression scores,  $t(392) = .98, p = .33$  differed over bipolar type. However, CGAS scores did differ,  $t(395) = -2.21, p = .03$ . Children diagnosed with Bipolar NOS scored higher on the CGAS ( $M = 56.57, SD = 11.41$ ) than children diagnosed with Bipolar I/II ( $M = 53.74, SD = 12.53$ ). Next, age of onset of bipolar symptoms was examined. The t-test was significant,  $t(399) = 2.21, p = .03$ . Children diagnosed with Bipolar NOS were younger at their diagnosis ( $M = 8.69, SD = 3.63$ ) compared to children diagnosed with Bipolar I/II ( $M = 9.60, SD = 4.05$ ).

Next t-tests were performed to consider differences in family functioning and bipolar type. Current family cohesion levels, as measured by the FACES-II Cohesion scale, current adaptability levels, as measured by the FACES-II Adaptability scale and conflict levels, as measured by the Conflict Behavior Questionnaire were indices of family functioning. Neither Cohesion levels,  $t(368) = -1.58, p = .12$ , adaptability levels,  $t(362) = -.13, p = .90$  nor conflict levels,  $t(383) = -1.17$  differed over bipolar type. Therefore differences were found between bipolar type in overall functioning and age of onset; no differences were found in family functioning or specific symptom presentation.

### *Differences by Sex*

Next, t-tests were performed to examine sex differences in symptom presentation. Mania rating scores did not differ for girls or boys,  $t(397) = -1.05, p = .30$ . However, depression scores did,  $t(393) = 3.13, p = .002$ . Girls with bipolar disorder were significantly more depressed ( $M = 16.86, SD = 11.98$ ) than boys ( $M = 13.63, SD = 9.33$ ). Sex was not related to CGAS scores,  $t(396) = .67, p = .50$ . Next, age of onset was examined. The t-test was significant,  $t(400) = -4.92, p < .001$ . Boys with bipolar disorder were found to be significantly younger ( $M = 8.41, SD = 3.70$ ) than females ( $M = 10.02, SD = 3.95$ ) at their time of diagnosis.

Further t-tests were performed to examine sex differences in family functioning for children with bipolar disorder. Neither cohesion levels,  $t(369) = 1.58, p = .12$  nor adaptability levels,  $t(363) = -.49, p = .62$  differed significantly. There was a marginal sex difference in family conflict with females ( $M = 8.56, SD = 5.11$ ) who had higher conflict scores than males ( $M = 7.72, SD = 4.32$ ), nor conflict levels,  $t(384) = -1.77, p = .08$ , differed over sex. Therefore, girls exhibit more depression than boys, but do not differ on other symptom measures. Additionally, there were no differences in family functioning between the sexes.

### *Differences by Pubertal Stage*

In order to examine the relationship between symptom presentation and pubertal stage t-tests were performed. Pubertal status was not related to mania rating scores,  $t(343) = 1.46, p = .15$ . It was related to depression scores,  $t(338) = -3.37, p = .001$ . Children who were post-pubertal scored higher ( $M = 16.58, SD = 10.90$ ) than children

who were pre-pubertal ( $M = 12.63$ ,  $SD = 8.28$ ) on depression. Finally, pubertal status was not related to scores on the CGAS,  $t(341) = -.67$ ,  $p = .50$ .

Additional t-tests were performed to examine pubertal differences in family functioning for children with bipolar disorder. Cohesion scores were related to pubertal stage,  $t(325) = 7.4$ ,  $p < .001$ . Families of pre-pubertal bipolar children scored higher on the cohesion scale ( $M = 65.23$ ,  $SD = 8.21$ ) than families of post-pubertal bipolar children ( $M = 56.44$ ,  $SD = 10.59$ ). Adaptability scores were also related to pubertal stage,  $t(321) = 2.39$ ,  $p = .02$ . Families of pre-pubertal bipolar children scored higher on the adaptability scale ( $M = 46.52$ ,  $SD = 6.17$ ) than families of post-pubertal bipolar children ( $M = 44.56$ ,  $SD = 7.13$ ). Additionally, conflict levels were also related to pubertal stage,  $t(339) = -2.69$ ,  $p = .007$ . Families of pre-pubertal bipolar children reported less conflict ( $M = 6.83$ ,  $SD = 4.09$ ) than families of post-pubertal bipolar children ( $M = 8.23$ ,  $SD = 4.59$ ). Thus, pubertal status was related to symptom presentation, with older children exhibiting more manic and depressive symptoms than younger children. Additionally, younger children tended to report more cohesion and adaptability within their families as well as less conflict.

#### *Relationships Between Symptoms and Family Functioning*

To explore the relationships between symptom presentation and family functioning over all bipolar diagnosis in the sample, simple correlations were first performed. Current mania symptom severity, depression symptom severity, and overall level of functioning, were indices of symptom presentation; family conflict, family cohesiveness and adaptability, were indices of family functioning. As shown in Table 2, mania symptom severity was positively correlated with family conflict. Depression



Table 2

*Relationships among symptoms, overall functioning and family functioning.*

	<b>Conflict</b>	<b>Cohesiveness</b>	<b>Adaptability</b>
Current MRS	.13*	-.07	-.04
Current Dep-P	.06	-.11*	-.08
<i>Current CGAS</i>	-.17**	.10	.04

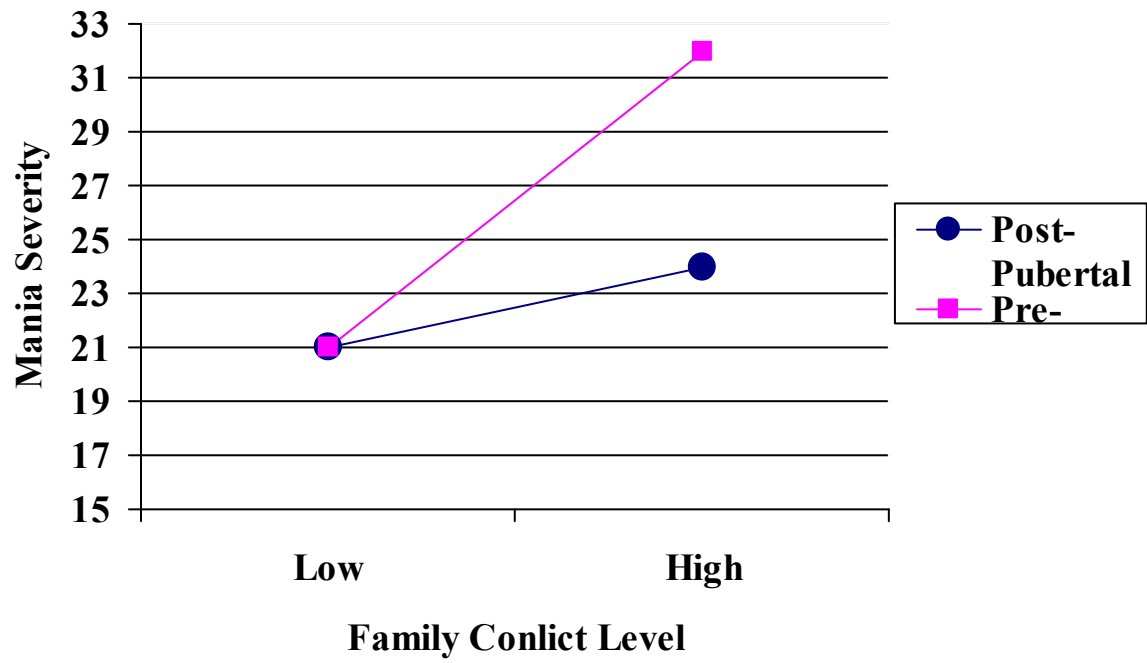
\*Correlation is significant at the 0.05 level (2-tailed)

\*\*Correlation is significant at the 0.01 level (2-tailed)

scores were negatively correlated with family cohesiveness and the overall level of functioning was negatively correlated with family conflict. Thus, family conflict was related to higher levels of mania and lower overall functioning, whereas family cohesion was related to lower levels of depression. These relationships were all quite modest.

*Conflict for Pubertal versus Pre-Pubertal Children.* Because research suggests that pre-pubertal mania differs from post-pubertal mania in important ways and because earlier t-tests suggested serial differences between pre and post-pubertal mania, we next examined whether the linear relationship between symptom presentation and family functioning was moderated by pubertal status. To test this, a series of multiple regressions was performed predicting symptoms from family functioning and pubertal status. First, a multiple regression analysis was performed predicting mania symptom severity from family conflict, pubertal status (entered as a dummy variable) and the interaction between the two. The regression was significant,  $F(3, 332) = 6.21, p < .001$ ,  $R^2 = .05$ . Family conflict ( $\beta = .40, p < .01$ ) and the interaction between conflict scores and pubertal status ( $\beta = -.39, p = .008$ ) made independent contributions to the prediction of MRS scores, but pubertal status alone did not. Thus, higher family conflict predicted higher mania symptoms in children, but this relationship was affected by pubertal status. As seen in Figure 1, the relationship between family conflict and mania severity was stronger for pre-pubertal children. A parallel regression was performed using depression symptom severity and was also shown to be significant  $F(3, 327) = 4.26, p = .006$ ,  $R^2 = .04$ . Pubertal status ( $\beta = .29, p = .008$ ) was a significant predictor of depression severity, but neither family conflict nor the interaction between family conflict and

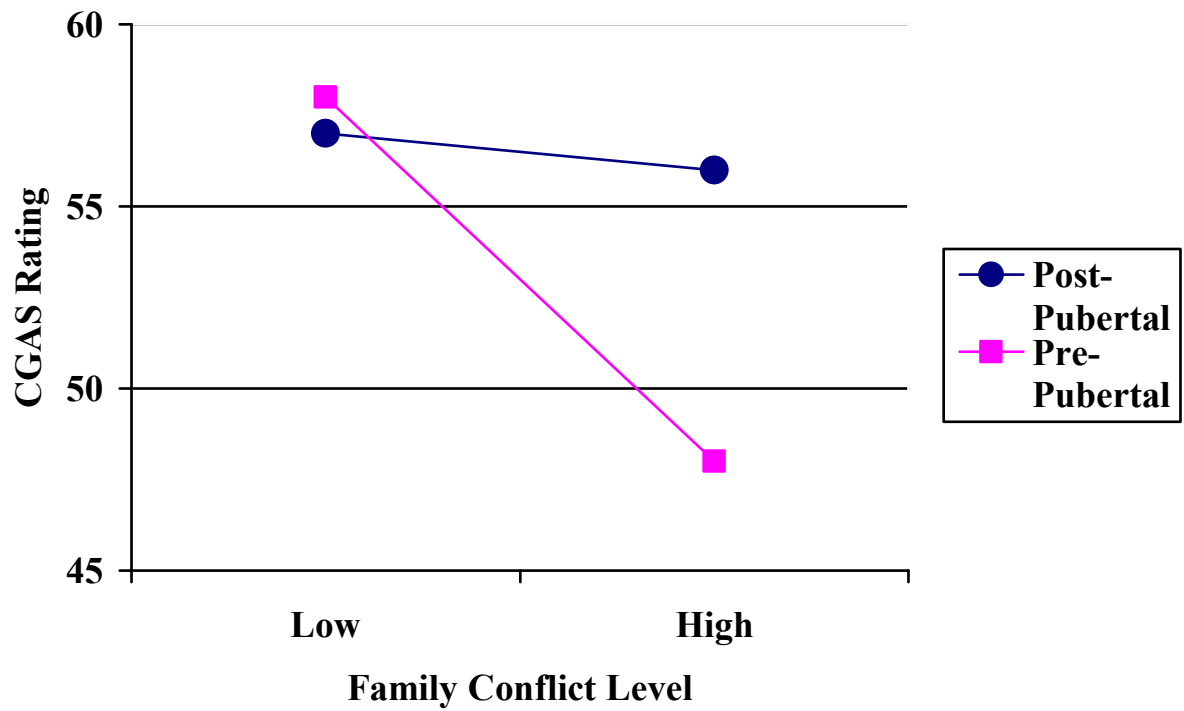
*Figure 1: The relationship between family conflict and mania symptom severity as moderated by pubertal status*



pubertal stage was significant in predicting depression level. As seen in earlier analyses, post-pubertal children had higher depression scores ( $M = 16.58$ ,  $SD = 10.90$ ) than pre-pubertal children ( $M = 12.63$ ,  $SD = 8.28$ ). Finally, a third regression with these same predictor variables was performed using overall functioning as the dependent variable. This regression was significant,  $F(3, 331) = 4.12$ ,  $p = .007$ ,  $R^2 = .04$ . Both family conflict ( $\beta = -.36$ ,  $p = .001$ ) and the interaction between family conflict and pubertal status ( $\beta = .40$ ,  $p = .008$ ) significantly predicted overall functioning, but pubertal status alone did not. As seen in Figure 2, higher conflict in families was related to lower overall functioning, but this relationship was much stronger for pre-pubertal children.

*Cohesion for Pubertal versus Pre-pubertal Children.* A second set of regression analyses was performed predicting symptoms and functioning from family cohesiveness. More research has examined conflict and its relationship to symptom severity but it is likely that other aspects of family functioning might also be related. First, mania symptoms were predicted from cohesiveness, pubertal status (entered as a dummy variable), and the interaction between the two. The overall regression was only marginally significant  $F(3, 33) = 2.35$ ,  $p = .07$ . A parallel regression was then performed on depression symptom severity. The regression was significant  $F(3, 329)$ ,  $p = .008$ ,  $R^2 = .04$ . However, none of the variables made a significant independent contribution to the prediction of depression severity. Finally, a third regression was performed on overall functioning. The overall regression was not significant  $F(3, 332) = 1.70$ ,  $p = .17$ . In contrast to family conflict, family cohesiveness was not as predictive of symptoms and child functioning, even when pubertal status was taken into account.

*Figure 2: The relationship between family conflict and child global assessment scale rating as moderated by pubertal status*



*Adaptability for Pubertal versus Pre-pubertal Children.* A third set of regression analyses was performed predicting symptoms and functioning from another aspect of family functioning, family adaptability. First, mania symptom severity was predicted from family adaptability, pubertal status (entered as a dummy variable) and the interaction between the two. The overall regression was not significant  $F(3, 332), p = .38$ . Another parallel regression was performed on depression severity. The regression was significant  $F(3, 329) = 3.90, p = .009, R^2 = .03$ . However, none of the variables made significant independent contributions. A final regression was performed using overall functioning as the dependent variable. The overall regression was not significant  $F(3, 332) = .31, p = .82$ . As with family cohesion, family adaptability was not a significant linear predictor of child symptoms and overall functioning, even when pubertal status was taken into account.

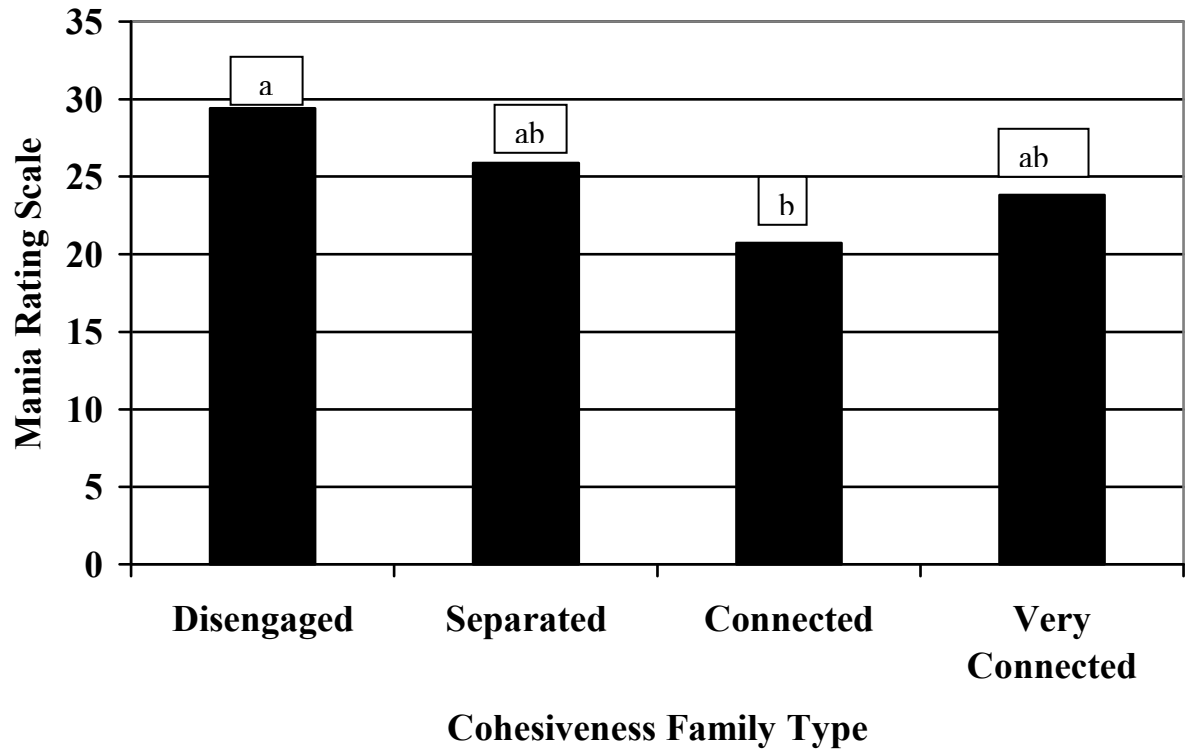
*Family Type and Symptom Severity.* Although measures of non-conflictual family functioning were not linearly related to symptoms and overall functioning, it may be that bipolar severity is better or worse for certain family types. Families can be divided into types based on their cohesiveness. These types include disengaged, separated, connected and very connected. The next analyses explore these different family types and the possibility of a nonlinear relationship between cohesion and symptom severity. A univariate analysis of variance (ANOVA) was performed examining the impact of family cohesiveness type and pubertal status on symptom presentation using the Mania Rating Scale. A 2 (puberty stage) by 4 (cohesive family type) model was used. The analysis revealed significant effects for cohesiveness type,  $F(3, 322) = 4.83, p = .003$ , as well as for pubertal status,  $F(1, 314) = 6.71, p = .01$ . The interaction between pubertal status

and cohesiveness was not significant,  $F(3, 322) = .99, p = .39$ . An examination of means revealed that post-pubertal children ( $M = 27.32, SD = 1.56$ ) scored higher on the mania rating scale than pre-pubertal children ( $M = 22.60, SD = .94$ ). Additionally, follow-up Tukey tests revealed significant differences between the mania scores of children in disengaged families versus connected families (see Figure 3). Significant differences were not found between the other family types.

A parallel ANOVA was performed using depression severity as the dependent variable. This test was not significant for cohesiveness,  $F(3, 317) = .78, p = .50$ , pubertal status,  $F(1, 317) = 3.21, p = .07$ , or the interaction between the two,  $F(3, 317) = .25, p = .87$ . Finally, a third ANOVA was performed using overall functioning as the dependent variable. This test was also not significant for cohesiveness,  $F(3, 312) = 1.58, p = .20$ , pubertal status,  $F(1, 312) = 1.36, p = .25$ , or the interaction between the two,  $F(3, 312) = 1.23, p = .30$ .

Just as the cohesiveness scale can be used to divide families into types, so can the adaptability scale. Along these lines, families can be divided into: rigid, structured, flexible and very flexible. An ANOVA was performed to examine the impact of family adaptability type and pubertal status on symptom presentation using the Mania Rating Scale. A 2 (pubertal status) by 4 (adaptability family type) model was used. This test revealed no significant effects for adaptability,  $F(3, 318) = 2.13, p = .10$ , pubertal status,  $F(1, 318) = .227, p = .13$ , or the interaction between the two,  $F(3, 32) = .30, p = .83$ . A second ANOVA was performed using the depression scale as the dependent variable for symptom presentation. The test revealed no significant effects for adaptability,  $F(3, 313) = .49, p = .69$ , pubertal status,  $F(1, 313) = 2.68, p = .10$ , or the interaction between the

Figure 3: The relationship between family cohesiveness type and mania symptom severity





two,  $F(3, 313) = .69, p = .56$ . A final parallel ANOVA was performed using the CGAS as a predictor for symptoms and dependent variable. There were no significant effects for adaptability,  $F(3, 316) = .51, p = .68$ , pubertal status,  $F(1, 316) = .79$ , or the interaction between the two,  $F(3, 316) = .35, p = .79$ . Thus, family type based on cohesion was more strongly related to bipolar severity than family type based on adaptability. However, only one difference was noted: Children in disengaged families were more manic than children in connected families.

## Discussion

This study was designed to examine symptom presentation in bipolar youth as well as the relationship between symptom severity and family functioning. Other factors such as bipolar type, sex, and pubertal status were also explored to examine their relationship with manic symptoms, depressive symptoms and overall functioning. The study found a small but significant overall relationship between some aspects of family functioning (especially family conflict) and symptom severity. Relationships between symptom severity and family functioning were stronger for pre-pubertal children. Family type analyses also clarified the relationship between some aspects of nonconflictual family functioning and bipolar symptom severity. Finally, descriptive analyses also found differences in symptom presentation by age, bipolar type and sex.

Few studies specifically investigate family conflict as a predictor of symptom severity in children with bipolar disorder. Within this study, greater levels of family conflict were related to higher levels of manic symptoms in children, which was hypothesized. The direction of the relationship between family conflict and mania symptoms cannot be determined from the present investigation. It is possible that the symptoms of the child create more conflict within the family, that family conflict creates or increases symptoms in the child, or that the relationship is bidirectional. The relationship is probably a complex mix of both with both symptoms and conflict affecting each other and making each other worse.

The relationship between conflict and symptom severity was also dependent on the child's age. Conflict was more closely related to mania severity in younger children than in older children. Additionally, family conflict was also related to overall

functioning in children. More conflict was related to lower functioning, but as with mania, the relationship was stronger for younger children. Possible explanations for these findings include the fact that younger children are more dependent on their families and might be more affected by family conflict than are older children who have more opportunities or abilities to escape or distance themselves from the situation.

Conflict was not the only way that family functioning was evaluated within this study. Non-conflictual family functioning was examined through measures of cohesiveness and adaptability. Linear relationships between nonconflictual family functioning and symptom severity were not found. But there was a relationship between family type, based on cohesiveness, and symptom severity. Specifically, there was a relationship between cohesiveness type and mania symptom severity. Although the four groups of cohesion were not all different from one another, children in “disengaged” families presented significantly more mania symptoms than children in “connected” families. The scoring of the LIFE-II does not claim to portray the extremely high categories of “enmeshed” on their scale, but it can be assumed that the category of “very connected” is similar to an enmeshed category. Thus, even though “connected” families do not score the highest on the measure of connectedness, this is probably the healthiest level of cohesiveness within families. “Connected” families are likely to be engaged but not over-involved in their child’s life. Connected families may be more likely to notice when their children begin to present with symptoms, and to do something productive about it. Conversely, in disengaged families, symptoms may continue and worsen because family members are not in tune with one another. In this situation, problems can continue and treatment may not be sought, at least not right away. Similarly, children in

disengaged families may need to act out more or escalate behaviors in order to catch attention of parents. Even though a relationship between cohesion family type and mania symptom severity was found, there was no similar relationship with depressive symptom severity. Family adaptability, the other measure of nonconflictual family functioning, was not related to symptom severity in any of the analyses.

Previous research supports the effects of family factors and their influence on relapse and re-hospitalization in adult bipolar patients (Holahan, & Moos, 1987; Milkowitz, et al., 1988; Rea, et al., 2003; Johnson & Roberts, 1995), but specific conflict behaviors, and other family factors, have not been examined extensively in children. Previous studies have noted that overall, families with more conflict are more likely to have a member with psychopathology, especially bipolar disorder (Chang, Blasey, Ketter, & Steiner, 2001). Past research has shown that families with bipolar disorder differ on family functioning compared to controls. Chang and colleagues (2001) reported that within families with a parent suffering from bipolar disorder, there is less cohesion when compared to control families. Other research on adults can be used to understand the relationship between family conflict and symptoms, and to make predictions about whether this relationship might also exist in childhood. A recent study by Christensen Gjerris, Larsen, Bendtsen, Larsen, Rolff, Ring & Schaumberg (2003; as cited in Hooley, Woodberry & Ferriter, 2005) found that high levels of conflict preceded the onset of a depressive episode in adult females with bipolar disorder.

Conflict as a factor in family functioning can generate stress for individual family members, and stress has been shown to influence the expression of both manic and depressive episodes (Johnson & Roberts, 1995). Although most studies have focused on

adults, these findings should also generalize to children, as stress from family conflict may affect a child more than an adult because of their lack of control in the situation. It is possible that although the current investigation did not find a relationship between concurrent family conflict and depressive symptoms, that family conflict may precede the onset of depression, or of mania, for children. Future longitudinal research will be needed to address questions like this.

Review studies specifically examining differences between older and younger children with bipolar disorder are sparse. In a study specifically examining younger children with psychopathology, Fristad and Clayton (1991) found that children with mood disorders had lower rates of family dysfunction when compared to children with other mental illnesses. Although the current study did not have a control group in which to compare, connections about family conflict and symptom severity are contradicted within this study. In examining differential symptom presentation by age, Bowring and Kovacs (1992) found that younger children express more “externalizing” disorders than older children, such as “acting out,” cheating in school or picking fights. The symptoms in mania may be seen as externalizing because of many of the common symptoms. This may be one explanation for the difference in symptom severity between older and younger children.

In addition to questions of family functioning, analyses of bipolar symptoms across age, bipolar type and sex were examined in this study. Analyses revealed that older children exhibited more depressive symptoms than younger children. This may be linked to the question about differential symptom classifiers for children depending on their developmental stage. The most likely explanation is that older children have more

classic depressive symptoms and the measure detects this. Also, younger children are more likely to present with hyperactive symptoms, more characteristic of mania than of depressive symptoms. While the depression scale used in this study is modeled after the diagnostic criterion specified for children, they may not take into account developmental differences found in very young children. Although the measure is approved for use in the diagnosis of children of all ages, differential behaviors that are more common in younger children may not be adequately accounted for. Interestingly enough, pubertal status was not related to mania symptoms or overall functioning in children despite the symptom severity differences for depression. Moreover, pubertal status was also related to family factors, including family conflict and cohesion levels. Families of younger children were significantly more cohesive than older children as well as experiencing less conflict. These two findings are probably interrelated because where cohesion levels are high, there is more communication, and probably less conflict.

Another important question that has been raised in the discussion about childhood bipolar disorder is the vague nature of the Bipolar NOS category. Confusion still remains over its exact presentation, course and other factors. Interestingly enough, this study found no significant differences between bipolar type and symptom presentation, with either manic or depressive episodes. This is interesting because both the mania scale and the depression scale are targeted toward the “classic” picture of mania and depression, which tend to look at typical mood episodes more central to Bipolar I or II. Contrary to one of the hypotheses of this study, children diagnosed with Bipolar NOS seem to be doing better in overall functioning than children diagnosed with Bipolar I or II. The study also revealed that children diagnosed with Bipolar NOS have

an earlier age of onset compared with children diagnosed with Bipolar I or II. The development of symptoms in the children with Bipolar NOS that meet diagnostic criterion for either Bipolar I or II is possible with age. Unfortunately, this study did not take into account what percentages of the children were originally diagnosed with Bipolar NOS at a younger age and then received a diagnosis of Bipolar I or II. These diagnostic timelines data were not part of the initial assessment. Future research following this sample over time would be able to address this question of change in diagnosis from Bipolar NOS to Bipolar I or Bipolar II.

Another important descriptive variable that was explored in this study was sex. In terms of manic symptom severity and overall functioning, no sex differences were found. There was, however, a difference in depression symptom presentation by sex. Consistent with the hypothesized sex difference, females reported more depressive symptoms than males. Additionally, there were no differences in family functioning and conflict by sex.

In reviewing the literature about these descriptive features of the disorder, many of the features explored touch on current issues in the diagnosis of the disorder in children. During the NIMH roundtable on Early-Onset Bipolar Disorder, questions about the differential diagnosis of Bipolar I/II versus Bipolar NOS were discussed. The researchers concluded that there were distinct differences in symptom presentation for children in these two categories. Specifically looking at Bipolar NOS, a worse and more chronic course is generally expected (NIMH, 2000). This contradicts findings in the current study which revealed higher overall functioning in children with Bipolar NOS compared to Bipolar I/II. This might be due to the fact that since children diagnosed with Bipolar NOS do not have “classic” bipolar disorder it is not as debilitating. The episodes

are not long or severe enough to constitute a diagnosis of Bipolar I or II and therefore do not cause as much disruption. In terms of age of onset by bipolar type, the researchers concluded that Bipolar NOS can be used as a “working diagnosis” for children who present with bipolar-like symptoms, and may go on to “develop” classic bipolar disorder with age (NIMH, 2000).

The roundtable also discussed symptom differences across puberty lines. The researchers concluded that there were differences between adolescents and young children, but did not recommend different measures to diagnose the disorder (NIMH, 2000). In terms of sex differences, most previous research has not noted differences within bipolar youth (Jerrell & Shugart, 2004; Biederman, Kwon, Wozniak, Mick, Markowitz, Fazio & Faraone, 2004). However, some studies note that females are more likely to present with depressive symptoms than manic symptoms (Biederman, et al., 2004). Additionally, other studies report that males are more likely to be younger at the time of diagnosis compared to females (Jerrell & Shugart, 2004) which was also found in the current investigation. This difference may be related to different symptom presentation that is less obvious in girls or the possibility that psychopathology in childhood is often overlooked in girls, resulting in later diagnosis (Silverthorn & Frick, 1999).

Childhood bipolar disorder is still very mysterious. More research is needed in order to understand this diagnosis in children. Because of this, there are many possibilities for future research. First, the relationship between family conflict and symptom severity needs to be examined further. The relationship between conflict and symptom severity found in this study was not very strong, but it is still important in



understanding its contribution to bipolar symptoms in children. In the present study, the utilization of an average conflict score, over all family members, provided a unique and more exact perspective on current conflict within the entire family system than either child or adult reports of family conflict alone. However, there may still be better ways of assessing family conflict that might show a stronger relationship with symptom severity. For example, expressed emotion has been an important measure of family conflict directed at bipolar patients and patients with other disorders. Not often studied in children expressed emotion assessed naturalistically, either from 5 minute speech samples where the number of spontaneous critical comments about the ill family member are counted, or from observations of lengthy observational family assessments.

It will also be important to understand the direction of the relationship between symptoms and family functioning. Measures of conflict and symptoms over time will be needed in future longitudinal research to assess how one influences the other. Tracking children at risk for bipolar disorder and assessing their symptoms and family functioning could also help clarify the relationships between conflict and bipolar symptoms. If conflict exists prior to the child's symptoms, or after general symptoms appear, but prior to the child's development of bipolar symptoms, researchers could be more confident about the causal relationship of family factors in the development of bipolar disorder.

Additional factors that need to be explored are cohesion levels within families. Since cohesion was seen as an important factor in symptom presentation, more research is needed to examine the differences in bipolar families. Since family therapy has been shown to be effective in children with bipolar disorder, understanding the protective properties of "connected" families would be important in determining how family factors

can be helpful to children. Additionally, understanding how to teach families to be more communicative, but not over-involved is vital.

Another finding in this study that should be explored further is the difference of symptom presentation by pubertal status. Since there was such a difference between children who were pre-pubertal versus those who were post-pubertal in symptom severity, a better understanding of the symptom differences is important. Because of the differences in both manic and depressive symptom severity, the development of a new symptom checklist for younger children may be necessary. Early-onset bipolar disorder maybe better understood as presenting different symptoms in different ages and thus requiring different symptom measures at different developmental stages.

Finally, sex differences have previously not been noted in children with bipolar disorder. Interestingly enough, this study revealed differences. Further research should examine these sex differences in children with bipolar disorder, in order [to see if differences in age of onset, and symptom presentation found in the present study replicate in other investigations, and to see if other sex differences exist, including differences in causes or course of illness. Additionally, investigating whether aspects of bipolar disorder differ jointly by age and sex would be useful. It may be that bipolar disorder in girls versus boys is similar in childhood, but becomes more different with age. Although the prevalence of bipolar disorder does not differ in adults by sex, men and women have a different course of illness, with more bipolar men having a chronic course with rapid cycling (Comer, 2005).

There were few limitations to this study, specifically because the data was obtained from a rigorous, federally funded study. Perhaps the biggest limitation was the

absence of a control group. Because of this, the current study was unable to examine family functioning in bipolar children compared to controls or even children with other psychological disorders. This would be useful in order to see whether the relationships that were found within this study pertaining to family factors are present in children without psychopathology or in children with other disorders. It would also have been helpful to look at different time periods and at symptom change depending on levels of conflict and family functioning. Since there were relationships between symptoms and family functioning, it would be interesting to see if the same types of relationships stand true during different time periods of either greater or lesser conflict within the same families.

An added limitation of this study was the self-report nature of the questionnaires. When filling out self-report questionnaires, people may report socially acceptable answers in order to look better for the researcher. Ideally, it would be useful to have an observational method of family functioning by research staff in order to gain an impartial assessment of functioning. Finally, since there was so much research on expressed emotion and its effects on symptom presentation, it would be important in future studies to obtain a measure of expressed emotion within the families, and relate that to both general family conflict and symptom severity in the children.

. Finally, an additional limitation of the current investigation is the large number of tests conducted, and the possibility of an elevated rate of Type I error. Multivariate statistics, like MANOVA, could have been helpful for some aspects of the investigation. MANOVA would have taken into account the correlations among dependent variables and better controlled for Type I error. MANOVAs were run for bipolar group, pubertal

status, and sex group comparisons on symptom type and family functioning, and the findings were quiet similar for pubertal status and sex group, but weaker for bipolar group. Therefore, most caution may be needed in the interpretation of these bipolar group differences.

There are so many questions about early-onset bipolar disorder that this study has only begun to scratch the surface. Further studies need to examine both the course and severity of symptoms in different ages and within different families in order to develop better treatment options for children suffering from this disorder. Even though the nature of bipolar disorder continues to generate controversy and is still debated within the psychological community, childhood manifestations of this disorder appear to be a real problem, even for younger children. Continued research to better understand its development and course in childhood will help improve the lives of the children and families who are suffering from the disorder.

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